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Case Report

A Case of Loss of Consciousness due to Epilepsy Diagnosed Using an Implantable Loop Recorder

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We report a case of clonic-tonic seizures diagnosed using an implantable loop recorder, a device for detecting cardiac arrhythmias. A 65-year-old man was referred to our hospital for loss of consciousness with myotonic jerks during sleep. He had experienced several similar episodes. No family history of sudden death was evident, and no structural heart disease was present. Coronary angiography with intracoronary acetylcholine (ACh) showed neither organic stenosis nor vasospastic angina. Ventricular tachyarrhythmias were not induced by programmed electrical stimuli. Sleep electroencephalography, brain magnetic resonance imaging and magnetic resonance angiography revealed no specific findings. We implanted a loop recorder to monitor rhythm abnormalities. One month later, an attack occurred at night. His wife recognized the episode and activated the implantable loop recorder. No arrhythmia was recorded, but myopotentials characteristic of tonic-clonic seizures were detected.
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Key words: Implantable loop recorder, Tonic-clonic seizure, Convulsive syncope, Epilepsy

Introduction

The implantable loop recorder (ILR) was introduced in Japan in 2009 for the diagnosis of heart rhythm disturbances. The most popular use of this device is for patients with recurrent unexplained syncope despite baseline work-up and complications due to falling. Several other applications have been described. Ho et al.[1)] reported the possibility of using this device to diagnose generalized tonic-clonic seizures, and we report an example herein.

Case Report

A 65-year-old man was referred to our hospital due to loss of consciousness (LOC) with myotonic jerks during sleep. Before the episode, he awoke at midnight to urinate and returned to bed. After 5 or 6 min, his wife noted that he was thrashing his arms

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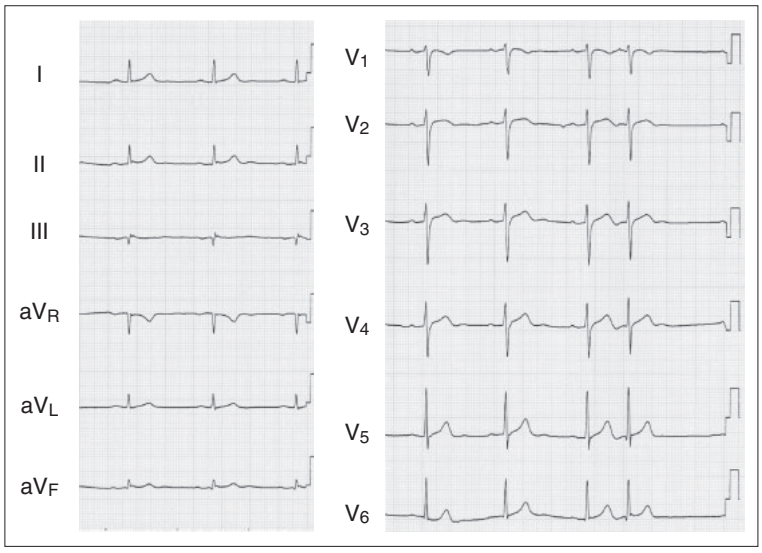

Figure 1 Surface 12-lead ECG.
Slight ST-segment elevation was seen in leads II, III and aV_F. The QT interval was normal.

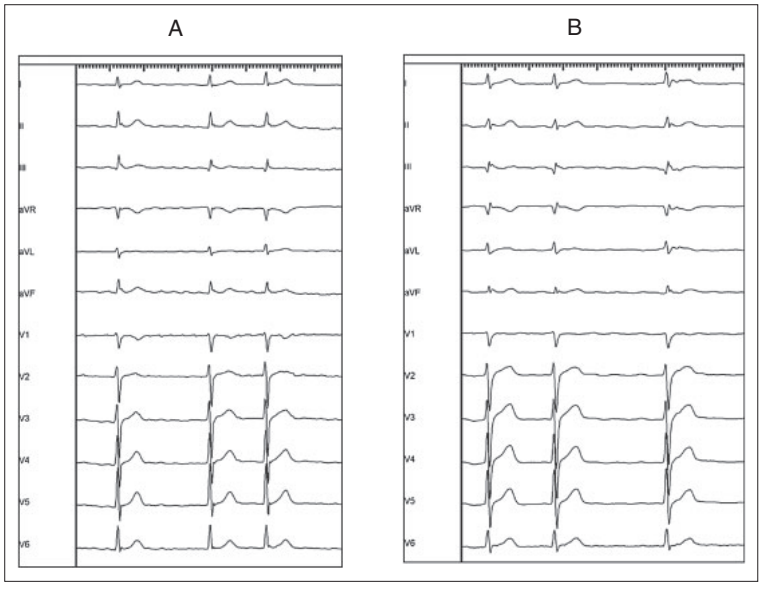

Figure 2 ECG before (**A**) and after (**B**) pilsicainide administration.
Rhythm changed to atrial fibrillation after acetylcholine injection during coronary angiography. ST-segment changes resembling Brugada syndrome were not seen in any leads.

and legs and rolling his eyes. He lost consciousness and did not react to slaps on the cheek. Consciousness was regained after a few minutes. He could talk within 10 min after the attack, but still showed mild disorientation. Similar episodes were noted during sleep on subsequent nights. No family history of sudden death was evident, and the patient had no past history of note. Electrocardiography (ECG) showed sinus rhythm with premature atrial contraction (**Figure 1**). Heart rate was 51 beats/min. PR interval was 0.20 s, QRS duration was 0.11 s and QT interval was 0.44 s. Q wave was recorded in lead III. The ST-segment in leads II, III and aV_F was slightly elevated. Echocardiography showed normal cardiac structure and function. Coronary angiography showed no coronary stenosis at baseline and 75% narrowing was induced in segment 1 of the RCA using intracoronary ACh 50 μg and in segment 7 of the LAD using intracoronary ACh 100 μg. However, he did not complain of chest pain and no ECG changes were observed. An electrophysiological study (EPS) showed that the HV interval was 40 msec. Sinus node function was not evaluated because atrial fibrillation was induced by intracoronary injection of ACh and sustained during EPS. Programmed electrical stimuli (PES) of triple extra

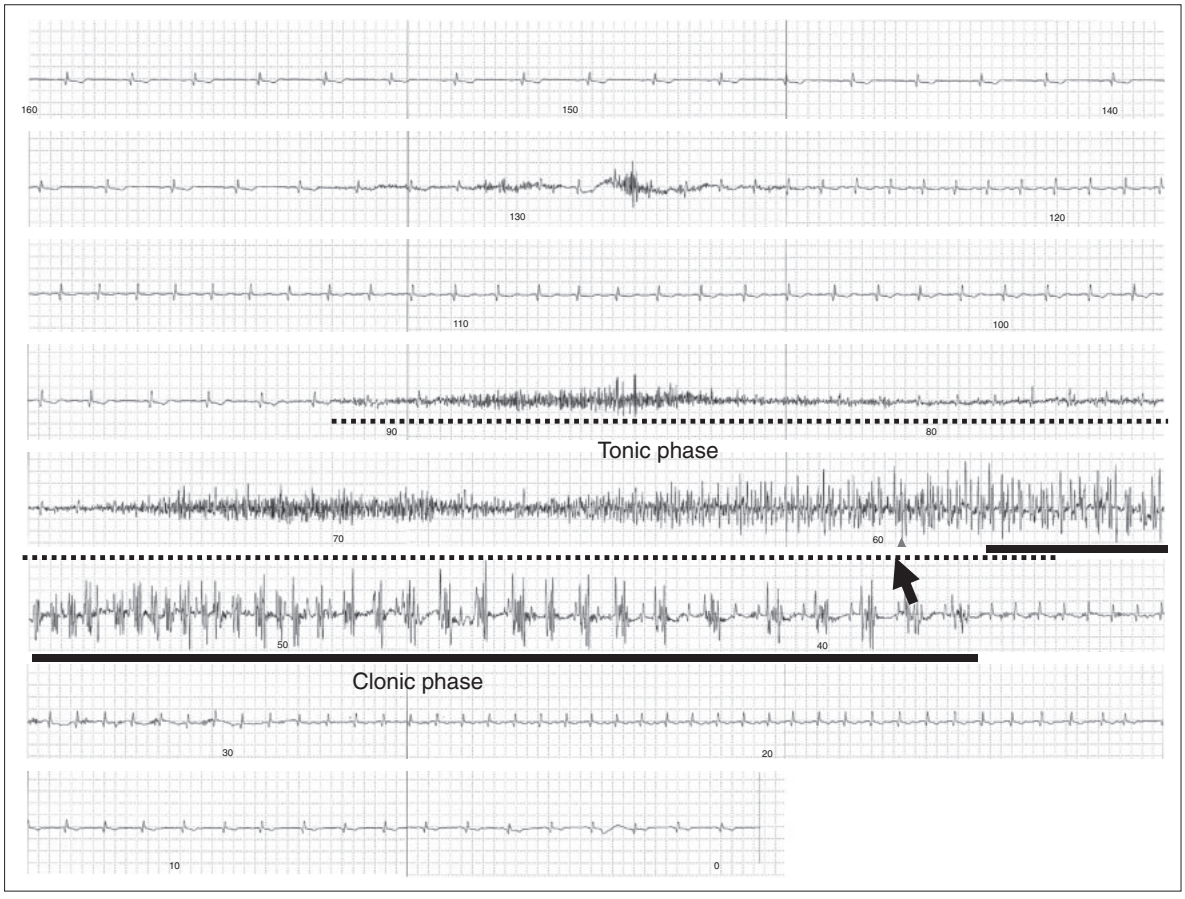

Figure 3 ECG recorded in the ILR.
ECG for 6 min before activation and 1 min after activation was stored in the ILR. Arrow indicates the time of activation. Background ECG was sinus rhythm at 60 beats/min. The dotted underline shows the tonic phase. Artifacts of sustained, rapid, high-frequency myopotentials covered the ECG for about 45 s. Periodic bursts of myopotentials from 3–6 Hz to 1–2 Hz were then recorded on background ECG and continued for about 30 s. These findings were consistent with the clonic phase (solid underline). Heart rate after the seizure was 140 beats/min (sinus tachycardia).

beats induced only 8 consecutive ventricular contractions. PES was repeated after intravenous administration of pilsicainide 100 mg. A Brugada-like ST segment change was not observed in any leads (**Figure 2**) and ventricular tachyarrhythmias were not induced. Sleep electroencephalography was performed twice, but no epileptic spikes were recorded. Overnight polysomnography also did not detect epileptic spikes, although mild sleep apnea was detected. Brain magnetic resonance imaging and magnetic resonance angiography did not show any specific and/or abnormal findings. We could not confirm the diagnosis after baseline work-up and further specific evaluations. The possibility of ventricular arrhythmia or bradycardia was not completely excluded. We therefore implanted a loop recorder (Reveal DX; Medtronic, Minneapolis) to monitor rhythm abnormalities. One month later, an attack occurred at night and was recognized by the patient's wife, who then activated the ILR. No arrhythmia was recorded during the episode, but myopotentials characteristics of tonic-clonic seizure were detected (**Figure 3**).

Discussion

LOC with convulsion is usually due to either seizures or syncope. However, differentiating between epilepsy and convulsive syncope is sometimes difficult. Several reports have described misdiagnosis of epilepsy.[2,3] Indeed, some studies have reported that 30–42% of patients initially thought to have epileptic seizures were later found to have convulsive syncope due to cardiovascular causes.[3,4] Many cardiovascular disorders may cause LOC complicated by abnormal movements attributable to general-

ized cerebral hypoxia. Grubb et al.[5] induced syncope with tonic-clonic seizure-like episodes in 67% of head-up tilt (HUT)-positive patients. They also reported 3 syncopal cases with convulsive activities for whom causes remained unknown even after extensive neurological and cardiovascular evaluations.[6] Those cases were diagnosed as neurocardiogenic syncope after prolonged monitoring with ILRs. All 3 patients showed long asystole. Two patients took the HUT test twice and one patient took the test once with no useful results. Ho et al.[1] implanted an ILR in patients with tonic-clonic seizures to evaluate the potential for further lethal arrhythmias, as epilepsy patients sometimes die suddenly. ILRs recorded sustained, rapid, high-frequency myopotentials identical to the tonic phase, then periodic bursts of high-frequency myopotentials with a decelerating burst frequency identical to the clonic phase. All recorded episodes were stereotypical. This means that the ILR is also useful to diagnose tonic-clonic seizures. However, the use of ILR for diagnosing generalized tonic-clonic seizure shows some limitations, as the rapid frequency of myopotential artifacts may exceed the nonprogrammable band-pass filter of the device. In addition, someone must activate the device, as no automatic capture function is available. Reveal DX can be programmed to record ECG from 6.5 min before patient activation and to 1 min after activation. The level of consciousness of epileptic patients after an attack is not clear and deteriorated consciousness often persists for a certain time. The limited recording time means that another individual must activate the device in most cases. Complex partial seizures and all generalized seizures including absence, myoclonic, clonic, tonic, tonic-clonic, and atonic seizures involved LOC. Tonic seizure should show myopotential artifacts in the tonic phase and clonic seizure show artifacts in the clonic phase, suggesting that these types of seizure may be diagnosed by ILR. However no such cases have been reported. We encountered a case showing LOC with convulsions of unknown origin. Intensive neurological and cardiovascular examinations were unable to confirm the cause. We could not completely exclude the possibility of convulsive syncope due to arrhythmia, even though provocation tests yielded negative results. We were also unable to confirm epilepsy, as neurological tests all provided negative results. LOC recurred and the patient and his wife were thus quite anxious. We decided to implant an ILR to exclude the possibility of potentially lethal arrhythmia. The ILR recognized progressive sinus tachycardia during LOC, which did not suggest certain mechanisms of syncope.[7] However, the ILR recorded typical myopotentials of tonic-clonic seizure, as Ho et al.[1] indicated. We conclude that ILR can be useful to identify causes of convulsive LOC when intensive neurological and cardiovascular evaluations prove inconclusive.

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